

KCNJ10 Antibody (Center)

Affinity Purified Rabbit Polyclonal Antibody (Pab) Catalog # AP17230c

Specification

KCNJ10 Antibody (Center) - Product Information

Application WB,E
Primary Accession P78508

Other Accession <u>P49655</u>, <u>Q9JM63</u>, <u>NP_002232.2</u>

Reactivity
Predicted
Host
Clonality
Isotype
Antigen Region

Human
Mouse, Rat
Rabbit
Polyclonal
Rabbit IgG
203-230

KCNJ10 Antibody (Center) - Additional Information

Gene ID 3766

Other Names

ATP-sensitive inward rectifier potassium channel 10, ATP-dependent inwardly rectifying potassium channel Kir41, Inward rectifier K(+) channel Kir12, Potassium channel, inwardly rectifying subfamily J member 10, KCNJ10

Target/Specificity

This KCNJ10 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 203-230 amino acids from the Central region of human KCNJ10.

Dilution

WB~~1:2000

E~~Use at an assay dependent concentration.

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

Storage

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

KCNJ10 Antibody (Center) is for research use only and not for use in diagnostic or therapeutic procedures.

KCNJ10 Antibody (Center) - Protein Information

Name KCNJ10 (HGNC:6256)



Function May be responsible for potassium buffering action of glial cells in the brain (By similarity). Inward rectifier potassium channels are characterized by a greater tendency to allow potassium to flow into the cell rather than out of it (PubMed:8995301). Their voltage dependence is regulated by the concentration of extracellular potassium; as external potassium is raised, the voltage range of the channel opening shifts to more positive voltages (PubMed:8995301). The inward rectification is mainly due to the blockage of outward current by internal magnesium. Can be blocked by extracellular barium and cesium (PubMed:8995301). In the kidney, together with KCNJ16, mediates basolateral K(+) recycling in distal tubules; this process is critical for Na(+) reabsorption at the tubules (PubMed:24561201).

Cellular Location

Membrane; Multi- pass membrane protein. Basolateral cell membrane. Note=In kidney distal convoluted tubules, located in the basolateral membrane where it colocalizes with KCNJ16.

Tissue Location

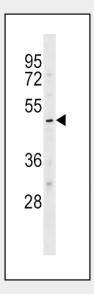
Expressed in kidney (at protein level) (PubMed:24561201). In the nephron, expressed in the distal convoluted tubule, the connecting tubule, the collecting duct and cortical thick ascending limbs (PubMed:20651251).

KCNJ10 Antibody (Center) - Protocols

Provided below are standard protocols that you may find useful for product applications.

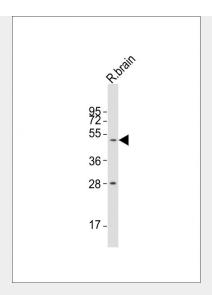
- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

KCNJ10 Antibody (Center) - Images



KCNJ10 Antibody (Center) (Cat. #AP17230c) western blot analysis in HL-60 cell line lysates (35ug/lane). This demonstrates the KCNJ10 antibody detected the KCNJ10 protein (arrow).





Anti-KCNJ10 Antibody (Center)at 1:2000 dilution + rat brain lysates Lysates/proteins at 20 μ g per lane. Secondary Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated at 1/10000 dilution Predicted band size : 43 kDa Blocking/Dilution buffer: 5% NFDM/TBST.

KCNJ10 Antibody (Center) - Background

This gene encodes a member of the inward rectifier-type potassium channel family, characterized by having a greater tendency to allow potassium to flow into, rather than out of, a cell. The encoded protein may form a heterodimer with another potassium channel protein and may be responsible for the potassium buffering action of glial cells in the brain. Mutations in this gene have been associated with seizure susceptibility of common idiopathic generalized epilepsy syndromes.

KCNJ10 Antibody (Center) - References

Jonard, L., et al. Int. J. Pediatr. Otorhinolaryngol. 74(9):1049-1053(2010) Reichold, M., et al. Proc. Natl. Acad. Sci. U.S.A. 107(32):14490-14495(2010) Heuser, K., et al. Epilepsy Res. 88(1):55-64(2010) Pawelczyk, M., et al. Ann. Hum. Genet. 73 (PT 4), 411-421 (2009): Yang, T., et al. Am. J. Hum. Genet. 84(5):651-657(2009)